WEST Search History

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DATE: Monday, April 04, 2005

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		DB=PGPB,USPT,EPAB,JPAB,DWPI; PLUR=YES; OP=OR	
☐	L11	L9 and (fusion or chimer$) same (heparan adj sulfate or heparan) same (GMCSF or GM-csf or (granulocyte adj (macrophage-stimulating or macrophage adj stimulating) or granulocyte-macrophage adj stimulating) adj factor)	1
☐	L10	L9 and (heparan adj sulfate or heparan) same (GMCSF or GM-csf or (granulocyte adj (macrophage-stimulating or macrophage adj stimulating) or granulocyte-macrophage adj stimulating) adj factor)	33
☐	L9	L7 and (GMCSF or GM-csf or (granulocyte adj (macrophage-stimulating or macrophage adj stimulating) or granulocyte-macrophage adj stimulating) adj factor)	158
☐	L8	L7 and GMCSF or GM-csf or (granulocyte adj (macrophage-stimulating or macrophage adj stimulating) or granulocyte-macrophage adj stimulating) adj factor	12962
☐	L7	(heparan or heparan adj sulfate) same growth adj factor	784
☐	L5	l2 not l1	41
☐	L4	l2 and (syndecan or syndecan-2 or syndecan adj 2)	6
☐	L3	L2 and l1	3
☐	L2	(heparan or heparan adj sulfate) near3 (binding adj (region or domain or protein or $5peptide)) and (fusion or chimer$)	44
☐	L1	(syndecan or syndecan-2 or syndecan adj 2) with (fusion or chimer$) and heparan adj sulfate	20

END OF SEARCH HISTORY

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Field: **Title/Abstract**

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- To combine searches use # before search number, e.g., #2 AND #6.
- Search numbers may not be continuous; all searches are represented.
- Click on query # to add to strategy

Search	Most Recent Queries	Time	Result
#6	Search **(proteoglycan or syndecan*) AND ("granulocyte macrophage stimulating factor" or "granulcyte-macrophage stimulating" or "granulocyte macrophage-stimulating" or GMCSF or GM-CSF)** Field: **Title/Abstract**	16:46:48	13
#4	Search **(heparan or "heparan sulfate") AND ("granulocyte macrophage stimulating factor" or "granulcyte-macrophage stimulating" or "granulocyte macrophage-stimulating" or GMCSF or GM-CSF)** Field: **Title/Abstract**	16:44:22	15
#2	Search **#1 AND (growth factor or GMCSF or GM-CSF)** Field: **Title/Abstract**	16:08:29	13
#1	Search **("heparan sulfate binding" or "heparan sulfate attachment" or syndecan or syndecan-2) AND (fusion or chimer*)** Field: **Title/Abstract**	15:56:23	66

Clear History

STN

FILE 'HOME' ENTERED AT 16:48:16 ON 04 APR 2005

L1 QUE (SYNDECAN OR SYNDECAN-4 OR SYNDECAN (4A) 4 OR PROTEOGLYCAN OR HEPARAN (A) SULFATE) AND (GROWTH ADJ FACTOR OR GM-CSF OR GMCSF OR (GRANULOCYTE (A) (MACROPHAGE (A) (COLONY OR COLONY-STIMULATING) OR MACROPHAGE-COLONY) OR GRANULOCYTE-MACROPHAGE (A) (COLONY OR COLONY-STIMULATING)))

L2 476 (PROTEOGLYCAN OR HEPARAN (A)(SULFATE (A) (BINDING OR ATTACHMENT) OR SULFATE-BINDING OR SULFATE-ATTACHMENT)) (S) (FUSION OR CIMER######)

L7 615 (PROTEOGLYCAN OR HEPARAN (A)(SULFATE (A) (BINDING OR ATTACHMENT) OR SULFATE-BINDING OR SULFATE-ATTACHMENT OR SYNDECAN?)) (S) (FUSION OR CHIMER######)

L8 2 L7 AND (GROWTH ADJ FACTOR OR GM-CSF OR GMCSF OR (GRANULOCYTE (A) (MACROPHAGE (A) (COLONY OR COLONY-STIMULATING) OR MACROPHAGE-COLONY) OR GRANULOCYTE-MACROPHAGE (A) (COLONY OR COLONY-STIMULATING)))

L9 61 L7 AND (SYNDECAN OR SYNDECAN-2 OR SYNDECAN (A) 2) (S) (FUSION OR CHIMER? OR FUSED)

(FILE 'HOME' ENTERED AT 16:48:16 ON 04 APR 2005)

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, ...' ENTERED AT 16:48:52 ON 04 APR 2005

SEA (SYNDECAN OR SYNDECAN-4 OR SYNDECAN (4A) 4 OR PROTEOGLYCAN

1	FILE ADISCTI
1	FILE AGRICOLA
1	FILE BIOENG
58	FILE BIOSIS
5	FILE BIOTECHABS
5	FILE BIOTECHDS
23	FILE BIOTECHNO
36	FILE CANCERLIT
106	FILE CAPLUS
1	FILE CONFSCI
1	FILE DDFU
3	FILE DISSABS
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32	FILE WPIDS
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L1 QUE (SYNDECAN OR SYNDECAN-4 OR SYNDECAN (4A) 4 OR PROTEOGLYCAN

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, CANCERLIT' ENTERED AT 16:57:23 ON 04 APR 2005

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L2          476 S (PROTEOGLYCAN OR HEPARAN (A)(SULFATE (A) (BINDING OR ATTACHME
L3          364 S L1
L4           67 S L1 AND PY<1994
L5           34 DUP REM L4 (33 DUPLICATES REMOVED)
L6           13 S L5 AND (CHIMER OR FUSION OR SYNDECAN? OR GMCSF OR GM-CSF)
L7          615 S (PROTEOGLYCAN OR HEPARAN (A)(SULFATE (A) (BINDING OR ATTACHME
L8            2 S L7   AND (GROWTH ADJ FACTOR OR GM-CSF OR GMCSF OR (GRANULOCYT
L9           61 S L7 AND (SYNDECAN OR SYNDECAN-2 OR SYNDECAN (A) 2) (S) (FUSIO
L10           9 S L9 NOT PY>1994
L11          32 DUP REM L9 (29 DUPLICATES REMOVED)
L12          27 S L11 NOT L10
L13         476 S L7 AND L2
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L6 ANSWER 1 OF 13 MEDLINE on STN
AN 93280251 MEDLINE
DN PubMed ID: 8505374
TI Cell membrane-associated **proteoglycans** mediate extramedullar myeloid proliferation in granulomatous inflammatory reactions to schistosome eggs.
AU Alvarez-Silva M; da Silva L C; Borojevic R
CS Departamento de Bioquimica, Instituto de Quimica, Universidade Federal de Rio de Janeiro, Brazil.
SO Journal of cell science, **(1993 Feb)** 104 (Pt 2) 477-84.
Journal code: 0052457. ISSN: 0021-9533.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199307
ED Entered STN: 19930716
Last Updated on STN: 19930716
Entered Medline: 19930706
AB In chronic murine schistosomiasis, extramedullar myelopoiesis was observed, with proliferation of myeloid cells in liver parenchyma and in periovular granulomas. We have studied the question of whether cells obtained from granulomatous connective tissue may act as myelopoietic stroma, supporting long-term myeloid proliferation. Primary cell lines (GR) were obtained in vitro from periovular granulomas, induced in mouse livers by Schistosoma mansoni infection. These cells were characterized as myofibroblasts, and represent liver connective tissue cells involved in fibro-granulomatous reactions. They were able to sustain survival and proliferation of the multipotent myeloid cell lines FDC-P1 and DA-1 (dependent on interleukin-3 and/or **granulocyte-macrophage colony stimulating** factor, **GM-CSF**) without the addition of exogenous growth factors. This stimulation was dependent upon myeloid cell attachment to the GR cell layer; GR cell-conditioned medium had no activity. Primary murine skin fibroblasts could not sustain myelopoiesis. The endogenous growth-factor was identified as **GM-CSF** by neutralization assays with monoclonal antibodies. The stimulation of myelopoiesis occurred also when GR cells had been fixed with glutardialdehyde. The observed stimulatory activity was dependent upon heparan sulphate **proteoglycans** (HSPGs) associated with GR cell membranes. It could be dislodged from the cell layer with heparin or a high salt buffer. Our results indicate a molecular interaction between endogenous growth-factor and HSPGs; this interaction may be responsible for the stabilization and presentation of growth factors in myelopoietic stromas, mediating extramedullar proliferation of myeloid cells in periovular granulomas.

L6 ANSWER 3 OF 13 MEDLINE on STN
AN 92062227 MEDLINE
DN PubMed ID: 1953822
TI **Granulocyte-macrophage colony-stimulating** factor augments neutrophil-mediated cartilage degradation and neutrophil adherence.
AU Kowanko I C; Ferrante A
CS Department of Immunology, Adelaide Children's Hospital, Australia.
SO Arthritis and rheumatism, **(1991 Nov)** 34 (11) 1452-60.
Journal code: 0370605. ISSN: 0004-3591.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199111
ED Entered STN: 19920124
Last Updated on STN: 19920124
Entered Medline: 19911125
AB **Granulocyte-macrophage colony-**

stimulating factor (**GM-CSF**) is produced in large quantities by synoviocytes in the inflamed arthritic joint and is known to be a neutrophil activator. Neutrophils predominate during acute flares of arthritis and are important mediators of cartilage destruction. In this investigation, we show that treatment of neutrophils with 10-1,000 units/ml of **GM-CSF** augments their ability to degrade cartilage **proteoglycan** in vitro. This was associated with increased neutrophil adherence to cartilage and increased release of oxygen-derived reactive species and granule enzymes in response to cartilage. Coating the cartilage with heat-aggregated human immunoglobulin G (AHG) enhanced both neutrophil adherence to the tissue and tissue degradation. **GM-CSF**, however, augmented these neutrophil effects independently of the presence of AHG. In contrast, neutrophil-mediated inhibition of **proteoglycan** synthesis was unaffected by **GM-CSF**.

L6 ANSWER 5 OF 13 MEDLINE on STN
AN 88330928 MEDLINE
DN PubMed ID: 2458354
TI Characterization of a human eosinophil **proteoglycan**, and augmentation of its biosynthesis and size by interleukin 3, interleukin 5, and **granulocyte/macrophage colony stimulating** factor.
AU Rothenberg M E; Pomerantz J L; Owen W F Jr; Avraham S; Soberman R J; Austen K F; Stevens R L
CS Department of Medicine, Harvard Medical School, Boston, Massachusetts.
NC AI-22531 (NIAID)
AI-22563 (NIAID)
AI-23401 (NIAID)
+
SO Journal of biological chemistry, **(1988 Sep 25)** 263 (27) 13901-8.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198810
ED Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19881019
AB Human eosinophils were cultured for up to 7 days in enriched medium in the absence or presence of recombinant human interleukin (IL) 3, mouse IL 5, or recombinant human **granulocyte/macrophage colony stimulating** factor (**GM-CSF**) and then were radiolabeled with [35S]sulfate to characterize their cell-associated **proteoglycans**. Freshly isolated eosinophils that were not exposed to any of these cytokines synthesized Mr approximately 80,000 Pronase-resistant 35S-labeled **proteoglycans** which contained Mr approximately 80,000 glycosaminoglycans. RNA blot analysis of total eosinophil RNA, probed with a cDNA that encodes a **proteoglycan** peptide core of the promyelocytic leukemia HL-60 cell, revealed that the mRNA which encodes the analogous molecule in eosinophils was approximately 1.3 kilobases, like that in HL-60 cells. When eosinophils were cultured for 1 day or longer in the presence of 10 pM IL 3, 1 pM IL 5, or 10 pM **GM-CSF**, the rates of [35S]sulfate incorporation were increased approximately 2-fold, and the cells synthesized Mr approximately 300,000 Pronase-resistant 35S-labeled **proteoglycans** which contained Mr approximately 30,000 35S-labeled glycosaminoglycans. Approximately 93% of the 35S-labeled glycosaminoglycans bound to the **proteoglycans** synthesized by noncytokine- and cytokine-treated eosinophils were susceptible to degradation by chondroitinase ABC. As assessed by high performance liquid chromatography, 6-16% of these chondroitinase ABC-generated 35S-labeled disaccharides were disulfated disaccharides derived from chondroitin sulfate E; the remainder were monosulfated disaccharides derived from chondroitin sulfate A. Utilizing **GM-CSF** as a model of the cytokines, it was demonstrated that the **GM-CSF**

-treated cells synthesized larger glycosaminoglycans onto beta-D-xyloside than the noncytokine-treated cells. Thus, IL 3, IL 5, and **GM-CSF** induce human eosinophils to augment **proteoglycan** biosynthesis by increasing the size of the newly synthesized **proteoglycans** and their individual chondroitin sulfate chains.

L6 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1993:552125 CAPLUS
DN 119:152125
TI Methods of controlling the proliferation of macrophages
IN Maki, Richard A.; Celada, Antonio
PA La Jolla Cancer Research Foundation, USA
SO PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9314782	A1	19930805	WO 1993-US998	19930128 <--
	W: AU, BB, BG, BR, CA, FI, HU, JP, KP, KR, LK, MG, MN, MW, NO, NZ, PL, RO, RU, SD				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, SN, TD, TG				
	AU 9336098	A1	19930901	AU 1993-36098	19930128 <--
PRAI	US 1992-827106	A	19920129		
	WO 1993-US998	A	19930128		

AB This invention provides a method for stimulating or inhibiting the proliferation of differentiated macrophages by modulating the amount or activity of TGF-β in contact with said differentiated macrophages, as by contact with an effective amount of M-CSF or **GM-CSF** and TGF-β. The invention further provides a method for treating an individual with a condition characterized by the overabundance or lack of adequate differentiated macrophages by modulating the amount or activity of TGF-β activity in macrophage-containing tissues of said individual, as by administering an effective amount of M-CSF or **GM-CSF** and TGF-β, or by contacting differentiated macrophages with an agent, such as an antibody or decorin, which suppresses the activity of TGF-β. Further, the invention provides methods for stimulating proliferation of immature macrophages by contact with TGF-β. Controlling the proliferation of differentiated macrophages is useful in the prevention, suppression, or treatment of various pathologies, e.g. infections caused by invasion of foreign microbes or conditions relating to cancer-causing or other disease-related antigens.

L6 ANSWER 8 OF 13 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
AN 1993:355789 BIOSIS
DN PREV199345039214
TI The **syndecans**, co-receptors for matrix and growth factors, are induced selectively by an activity in wound fluid.
AU Gallo, R. L. [Reprint author]; Povsic, T.; Kim, C.; Page, C.; Eriksson, E.; Bernfield, M.
CS MGH/Harvard Cutaneous Biol. Res. Center, Boston, MA, USA
SO Journal of Investigative Dermatology, (1993) Vol. 100, No. 4, pp. 506.
Meeting Info.: Annual Meeting of the Society for Investigative Dermatology. Washington, D.C., USA. April 28-May 1, 1993.
CODEN: JIDEAE. ISSN: 0022-202X.
DT Conference; (Meeting)
LA English
ED Entered STN: 31 Jul 1993
Last Updated on STN: 3 Jan 1995

L6 ANSWER 10 OF 13 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
AN 1988:321849 BIOSIS
DN PREV198835027183; BR35:27183
TI REGULATION OF **PROTEOGLYCAN** PG BIOSYNTHESIS IN HUMAN EOSINOPHILS

EO BY INTERLEUKIN IL-3 IL-5 AND **GRANULOCYTE MACROPHAGE -COLONY STIMULATING** FACTOR **GM-CSF**.
AU POMERANTZ J L [Reprint author]; ROTHENBERG M E; OWEN W F; AUSTEN K F; STEVENS R L
CS HARV MED SCH, BOSTON, MASS 02115, USA
SO FASEB Journal, (1988) Vol. 2, No. 6, pp. ABSTRACT 8035.
Meeting Info.: 72ND ANNUAL MEETING OF THE FEDERATION OF AMERICAN SOCIETIES FOR EXPERIMENTAL BIOLOGY, LAS VEGAS, NEVADA, USA, MAY 1-5, 1988. FASEB (FED AM SOC EXP BIOL) J.
CODEN: FAJOEC. ISSN: 0892-6638.
DT Conference; (Meeting)
FS BR
LA ENGLISH
ED Entered STN: 11 Jul 1988
Last Updated on
ANSWER 13 OF 13 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
AN 88:191723 SCISEARCH
GA The Genuine Article (R) Number: M5985
TI REGULATION OF **PROTEOGLYCAN** (PG) BIOSYNTHESIS IN HUMAN EOSINOPHILS (EO) BY INTERLEUKIN (IL)-3, IL-5, AND **GRANULOCYTE MACROPHAGE-COLONY STIMULATING** FACTOR (**GM-CSF**)
AU POMERANTZ J L (Reprint); ROTHENBERG M E; OWEN W F; AUSTEN K F; STEVENS R L
CS HARVARD UNIV, SCH MED, BOSTON, MA, 02115
CYA USA
SO FASEB JOURNAL, (**1988**) Vol. 2, No. 6, pp. A1679.
DT Conference; Journal
LA ENGLISH
REC No References

L8 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:78268 CAPLUS
DN 134:146376
TI Fc fusion proteins for enhancing the immunogenicity of protein and peptide antigens
IN Gillies, Stephen D.; Lo, Kin Ming; Wesolowski, John S., Jr.
PA Lexigen Pharmaceuticals Corp., USA
SO PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001007081	A1	20010201	WO 2000-US19816	20000721
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2378866	AA	20010201	CA 2000-2378866	20000721
	EP 1198250	A1	20020424	EP 2000-950483	20000721
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
	BR 2000012569	A	20020528	BR 2000-12569	20000721
	JP 2003505431	T2	20030212	JP 2001-511964	20000721
	AU 779388	B2	20050120	AU 2000-63583	20000721
	RU 2248214	C2	20050320	RU 2002-104700	20000721
	NO 2002000255	A	20020315	NO 2002-255	20020117
	ZA 2002000501	A	20030121	ZA 2002-501	20020121
PRAI	US 1999-144965P	P	19990721		
	WO 2000-US19816	W	20000721		

AB Disclosed herein are methods and compns. for enhancing the immunogenicity of a preselected protein or peptide antigen in a mammal. Immunogenicity is enhanced by fusing the preselected antigen to an Ig heavy chain constant region to produce an Fc-antigen fusion protein. The Fc-antigen fusion proteins bind Fc receptors on the surface of antigen presenting cells, thereby targeting the antigen to the antigen presenting cells in the mammal. In addition, disclosed is a family of adjuvants, for example, an Fc-adjuvant fusion protein, for use in combination with the Fc-antigen fusion proteins to enhance or modulate a particular immune response against the preselected antigen.
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:487222 CAPLUS
DN 131:120861
TI Artificial proteoglycans for drug targeting and other therapeutic applications
IN Bennett, Kelly L.; Wolff, Edith A.; Aruffo, Alejandro A.; Greenfield, W. Brad
PA Bristol-Myers Squibb Company, USA
SO PCT Int. Appl., 61 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9937317	A1	19990729	WO 1999-US1411	19990121
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HU, ID, IL, IS, JP, KE, KG, KP,				

KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO,
NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA,
UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES,
FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI,
CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 9934488	A1	19990809	AU 1999-34488	19990121
US 6559287	B1	20030506	US 1999-235230	19990121
PRAI US 1998-72416P	P	19980124		
WO 1999-US1411	W	19990121		

AB Novel artificial proteoglycans containing a GAG assembly site and a control sequence required for assembly, method for enhancing the biol. activity of a glycosaminoglycan-binding protein using artificial proteoglycans, DNA constructs of artificial proteoglycans. The artificial proteoglycans of the present invention are useful for prepns. of adjuvants for vaccination, for targeting of chemokines to non-immunogenic tumor cells to enhance cellular anti-tumor response, for prepns. designed to help promote wound healing, and for treatment of immunol. disorders including rheumatoid arthritis, asthma, chronic obstructive pulmonary disorder, Lupus, inflammatory bowel disease, psoriasis, osteoarthritis, and HIV infection.

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 4 OF 9 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 94232974 EMBASE
DN 1994232974
TI Amino acid determinants that drive heparan sulfate assembly in a proteoglycan.
AU Zhang L.; Esko J.D.
CS Biochemistry/Molec. Genetics Dept., Schools of Medicine and Dentistry, University of Alabama,Birmingham, AL 35294, United States
SO Journal of Biological Chemistry, (1994) Vol. 269, No. 30, pp. 19295-19299. ISSN: 0021-9258 CODEN: JBCHA3
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 940826
Last Updated on STN: 940826
AB To study how cells regulate the composition of glycosaminoglycan chains on **proteoglycans**, we have examined the assembly of chains on **chimeric proteoglycans** containing segments of betaglycan (transforming growth factor- β Type III receptors) fused to protein A. Transient expression of the chimeras in Chinese hamster ovary cells revealed that only two glycosaminoglycan attachment sites exist. One site at Ser535 supported both chondroitin sulfate and heparan sulfate synthesis, whereas the site at Ser546 supported only chondroitin sulfate. The compositions of the chimeras were the same in CHO-K1, CHOP-C4, BHK-21, and HeLa S3 cells and in chimeras containing polyhistidine fused to the C terminus. Deletion experiments showed that the assembly of heparan sulfate chains on the chimeras required a peptide segment of ≤16 amino acids (SPGDSS535- GWPDGYEDLE) and the first 5 amino acids were not essential. Truncation of the acidic cluster (EDLE), site-directed mutation of the acidic residues in the cluster, or deletion of the sequence between the cluster and the Ser attachment site decreased heparan sulfate assembly. Mutation of Trp537 adjacent to the site also decreased heparan sulfate assembly. More importantly, introducing tryptophan next to three different Ser-Gly dipeptides in betaglycan and **syndecan -1 chimeras** stimulated assembly of heparan sulfate. Thus, one type of heparan sulfate attachment site consists of a Ser-Gly dipeptide and a flanking cluster of acidic residues. An adjacent tryptophan residue can augment the proportion of heparan sulfate.